"A COMPARATIVE STUDY OF LIPID PROFILE IN PREECLAMPSIA AND NORMOTENSIVE PREGNANT AND NONPREGNANT WOMEN"

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BONAFIDE CERTIFICATE

Certified that the dissertation titled "**A COMPARATIVE STUDY OF LIPID PROFILE IN PREECLAMPSIA AND NORMOTENSIVE PREGNANT AND NONPREGNANT WOMEN"** is a bonafide work done by Dr. M. MURALI KRISHNAN., under my guidance and supervision, in partial fulfillment of regulations of The Tamilnadu Dr. MGR Medical University for the award of M.D. Degree Branch I, (General Medicine) during the academic period from May 2010 to April 2013.

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DECLARATION

I solemnly declare that the dissertation titled **"A COMPARATIVE STUDY OF LIPID PROFILE IN PREECLAMPSIA AND NORMOTENSIVE PREGNANT AND NONPREGNANT WOMEN"** was done by me at K.A.P.V Government Medical College, Tiruchirappalli under the guidance and supervision **Prof. K.PARIMALADEVI.M.D.** The dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfillment of the requirement for the award of M.D. Degree in General Medicine.

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CONTENTS

INTRODUCTION

INTRODUCTION

Pre-eclampsia (PE), a non-convulsive form of hypertensive disorders complicating pregnanacy, accounts for a significant proportion of morbidity as well as death in both mother & foetus.[1]

The incidence of preeclampsia ranges from 5-15%. In India the prevalence rate of preeclampsia has been found out to be 8-10% of total number of pregnancies. The incidence in primigravidae is about 10% and in multigravidae about 5%(2). Based on the norms of American college of obstetrics & Gynecologist, the diagnostic criteria for hypertensive disorders complicating pregnancy is a) The value of systolic Blood pressure being more than or equal to140 mmHg b) the value of diastolic pressure being more than or equal to 90 mmHg Or 3) a rise of $\geq$ 30 mm/Hg in the value of systolic pressure Or 4) increase of $\geq$ 15 mmHg in Diastolic pressure, recorded in a female whose prior blood pressure has been normal .

It has been noted that the preeclampsia generally begins after 20weeks of gestation.A rise in blood pressure of more than 140/90mmhg along with proteinuria (either twenty four hour urine protein value being more than 300mg or a dipstick test which is positive with a value of 30mg/dl) is considered as the definition for preeclampsia. Preeclampsia is

characterized by vasospasam,increased peripheral vascular resistance,and reduced organ perfusion. The essential pathophysiology of preeclampsia has been noted as the elevated blood pressure which is resulting from the vasospastic mechanism occuring in human organs such as kidney, pregnant uterus, placenta & brain. Abnormalities of lipid production could result in a low ratio of PGI2: TXA2[3]. This has also been considered as one of the significant mechanism of increased blood pressure occuring in pregnancy. Alteration in lipid production and its metabolic pathway has a prominent role in hypertensive disorders complicating pregnancy

Inspite of the on going research works,the causative factors responsible for the incidence of preeclampsia could not be identified properly. Moreover, it has not been possible to find out a single test that can identify the females who are prone for developing preeclampsia[4]. The incidence of preeclampsia has been observed to be higher if abnormal lipid metabolism occurs in initial phase of gestation Lipid profile in females having a history of preeclampsia is found out to significantly distinct from that of females with normal gestation[5]. Abnormalities in the lipid metabolic pathway have been observed as an important factor resulting in the increased blood pressure & proteinuria that occur in preeclampsia. Disorders of lipoprotein metabolism are

reported to be a major cause of hypertension and proteinuria in Pre-eclampsia[6,7,8]. Inview of the above mentioned observations,it has been suggested that the abnormalities of lipid synthesis and its metabolic pathway might play a significant part in the occurence of features of Pre-eclampsia and Eclampsia. Our study has been carried out to understand the various abnormalities of lipid parameters in pregnant mothers presenting with pre-eclampsia.

AIMS AND OBJECTIVES

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1. To study the lipid profile in pre eclampsia
2. To explore the possibility of dyslipidemia in the pathogenesis of preeclampsia.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

PLASMA LIPIDS AND LIPOPROTEIN METABOLISM

Plasma Lipids

Lipids are seen in the following forms:

1. Fatty acids - straight chain compounds of varying lengths.
2. Triglycerides - consist of glycerol and 3 fatty acids.
3. Phospholipids - complex lipids, similar to TG but containing phosphate and a nitrogenous base.

The major phospholipids seen in plasma are lecithin and sphingomyelin. The phosphates and nitrogenous bases are soluble in water, which is an important quality for lipid transport. Cholesterol is having steroid structure, and all the other steroids are derived from it. In plasma 2/3 rd of the cholesterol exist esterified with FA as cholesterol esters.

LIPOPROTEINS

Lipids are partially soluble in water, to be carried in the body fluids they complexes with protein to form water soluble lipoprotein.

The water soluble part of protein, phospholipids and free cholesterol enclose a core of insoluble cholesterol esters and triglycerides. They are classified according to their densities into four main types.

Two are important for cholesterol transport :

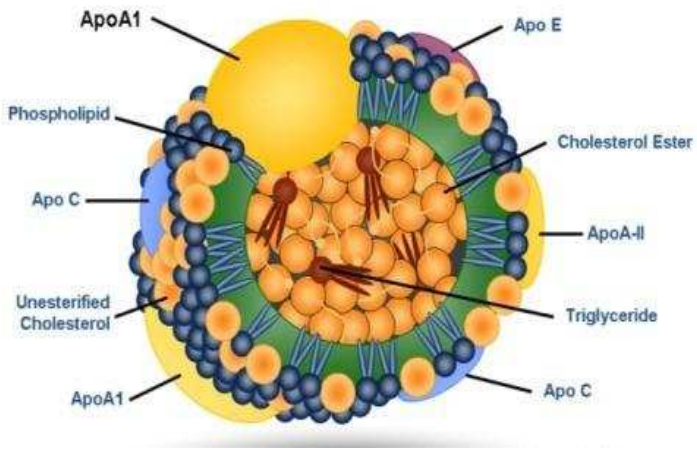
1. HDL (High density lipoprotein) which transports cholesterol from peripheral tissues.
2. LDL (Low density lipoprotein) transports cholesterol to peripheral tissues.

Other Two groups are important for TGL transport:

- VLDL (Very Low Density Lipoproteins) - transport endogenous triglyceride from the liver to peripheral tissues.
- Chylomicrons transport exogenous (dietary) triglycerides from gut to the liver.

A 5th Class also exist, IDL (Intermediate Density Lipoprotein) which is a transient intermediate in the metabolism of VLDL to LDL .

Plasma taken from a fasting subject contains only HDL, LDL and VLDL.



STRUCTURE OF LIPOPROTEIN

Chylomicrons

Chylomicrons serve as the carrier for the transport of dietary lipids in the plasma. They are synthesised iniside the enterocytes from the packaging of fat droplets .they are rich in triglycerides. When they are formed they contain onlyI apoB-48, apoA-I, apoA-II, and apoA-IV but later apo-C and apo- E are added from HDL. Their function is to transport dietary triglycerides from intestine to the adipose tissues for storage and to muscle and heart for their energy requirements. This exchange with HDL is important for subsequent lipolysis of chylomicrons by lipoprotein lipase (LPL) as apoE is needed for chylomicron binding to the endothelial surface and apoC-II is essential for activation of LPL[(2).] When mature chylomicron reaches the capillary networks perfusing the muscle and adipose tissues, they bind with endothelial surface through their apo-E. This binding result in interaction of chylomicrons with the endothelial LPL and its activation by apoC-II. LPL causes hydrolysis of the triglyceride content of chylomicrons and release of free fatty acids inside the capillaries. The major part of fatty acids released, diffuse into the adjacent myocytes for energy production or into adipocytes for storage[·(2)] The remaining part is called as chylomicron remnants, apoC and apoE exchanged back to HDL before their removal from the circulation by the liver and other tissues via LDL receptor-related protein (LRP).

VLDL, IDL, and LDL

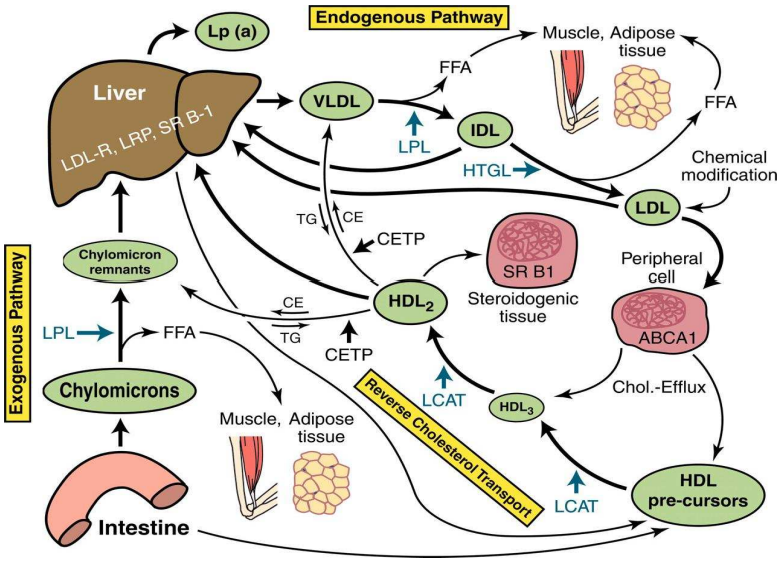
VLDL particles are synthesized by the liver and serve as the vehicle for transport of endogenous lipids to the peripheral tissues. Nascent VLDL is formed within the liver cells from the fusion of apoB-100 with a triglyceride-rich lipid droplet, followed by addition of apoE, apoA-I and apoA-II. After its release into the circulation, VLDL acquires apoC and apoE from HDL-2 in exchange for apoA-I, apoA-II, and phospholipids.

As similar to chylomicrons, this exchange with HDL-2 is essential for further metabolism of VLDL by LPL and the VLDL receptor. When circulaying through the capillaries perfusing the muscle and adipose tissues, VLDL particles attach to the endothelial surface via their apoE. This binding result in interaction of VLDL with LPL and the VLDL receptor. Following this apoC-II content of VLDL activate LPL, which causes hydrolysis of VLDL.

HDL

The most important function of HDL is retrieval and transport of cholesterol from the extrahepatic tissues for disposal in the liver[9]. This process which is commonly called as reverse cholesterol transport. This is very much important for cellular cholesterol homeostasis as well as

protection against atherosclerosis, kidney disease, and other complications. Other than this, HDL plays a major role in metabolism of triglyceride-rich lipoproteins by acting as a donor and acceptor of apoC and apoE for the metabolism chylomicrons and VLDL, a process which is essential in triglyceride metabolisam. Other funtions of HDL are, acting as potent endogenous inhibitor of inflammation, platelet adhesion, and LDL oxidation.



SUMMARY OF LIPID METABOLISAM

PREGNANCY HYPERTENSION

Hypertensive disorders of pregnancy tends to complicate nearly five percentage to ten percentage of total pregnant cases.

Along with hemorrhage & infection, these disorders constitute one limb of the dangerous triad of maternal morbidity as well as mortality. With hypertension, the *preeclampsia syndrome* , as been proven to be the most deadly one.

Elevated blood pressure seen in pregnancy which is otherwise known as gestational hypertension constitutes the non proteinuric increase in blood pressure. The study done by Berg & colleagues has suggested that nearly 50 percentage of the mortality that is occuring as a consequence of increased blood pressure could have been prevented.

NEW TERMINOLOGY AND CLASSIFICATION

1. Gestational hypertension—formerly called as pregnancy induced hypertension disorder
2. Preeclampsia (PE) and eclampsia syndrome
3. Preeclampsia syndrome superimposed on chronic hypertension
4. Chronic hypertension.

Gestational Hypertension

- Systolic Blood Pressure of140mmhg or diastolic Blood Pressure of 90 mm Hg , detected for the 1st time in pregnancy
- absence of excretion of protein in urine
- Blood pressure falling back to reference level prior to twelve weeks post delivery
- Only after the delivery, an ultimate diagnosis can be established.
- Patient might develop other Characteristic features of preeclampsia, like, pain and sensation of discomfort in the region of epigastrium or decrease in platelet count.

Preeclampsia (PE)

The following are the minimum criteria required for establishing a diagnosis.[4]

- Blood Pressure level of 140/90 mmHg after twenty wks of conception
- Twenty four hour urine protein excretion being 300mg or a dipstick assay giving the value of 1+.

The diagnosis can be made with a high index of certainity if the following criterias are fulfilled

- Blood pressure level of 160/110 mm Hg

- Twenty four hour urine protein excretion being 2gram or a dipstick assay giving the value of 2+.
- The creatinine level in the blood being more than 1.2 mg/dL
- Platelets count < 100,000/L
- Increased LDH suggestive of Microangiopathic hemolysis
- Increased values of liver enzymes such as elevated SGOT as well as SGPT
- Development of symptoms like headache or defects of vision. Continuous painful sensation in the region of epigastrium

Eclampsia

- A pregnant mother developing epilepsy which could not be explained by any other cause.

Preeclampsia which is being superimposed on a state of chronically elevated blood pressure

- Twenty four hour urine protein excretion being 300 mg which is of recent origin in a female who is a known case of hypertension without showing any excretion of protein in her urine prior to twenty wks of her pregnancy.

Rapid elevation in the quantity of protein which is being excreted in the urine or a rapid increase in BP or thrombocytopenia with a value of

platelet < 1 Lakh/dl developing in a female who has history of elevated BP & protein excretion in urine even prior to twenty wks of pregnancy.

Chronic Hypertension

- Females with a blood pressure in the range of 140/90mmHg which is being diagnosed either prior to conception or prior to twenty wks of conception and which could not be attributed to the trophoblastic disoders occuring in pregnancy.

or

- An elevated blood pressure which is being diagnosed for the first time after a period of twenty wks of gestation & the elevation being continuously present even after twelve wks of delivery.

RISK FACTORS

1. Young and nulliparous women

RECENT STUDIES - Sibai and Cunningham (2009),[4] found that the incidence of PE in nulliparous ranges from 3 to 10 percent, it is less than that for nulliparas. however, Ananth and Basso (2009) reported that the stillbirths were more commonly seen with in hypertensive multipara.

2. Obesity

3. Multifetal gestation,

4. Maternal age older than 35 years,

5. African-American ethnicity

6. Maternal obesity Body mass index (BMI)

Risk of PE increases from 4.3 % for women having a body mass index < 20 kilogram per metre square to 13.3 % in women having a BMI > 35 kilogram per metre square (Sibai and co-workers, 2000).

Follwing factors are associated with reduced risk of PE as shown by various studies

Smoking (Bainbridge and associates, 2005)

Placenta previa (Ananth and colleagues, 1997).

Normotensive during her first pregnancy (Getahun and colleagues 2007).

PATHOPHYSIOLOGY

The pathophysiology behind preeclampsia is unknown. Several theories have been postulated , but none of the theory point towards a single etiology. Understanding of the same can greatly influence the management and outcome.

Placental factor

The current concept is that the placenta plays an important role. Most of the recent studies support the theory of incomplete trophoblastic invasion of uterine spiral arteries, leading to reduced perfusion of

placenta, which causes production of antiangiogenic factors causing endothelial dysfunction. This abnormality of endothelium leads to the elevation in blood pressure, altered permeability of vasculature system well as the stimulation of coagulation pathway. It also cause damage to filtering mechanism in the kidney leading to proteinuria. This circulating angiogenic factors are demonstrated in serum of preeclapsic patients even before the disorder has shown manifestations clinically. Eventhough the above mentioned pathogenitic mechanism is very much appealing, it is not accepted by the foundation for pre-eclampsia. This was according to the study done by Ness et al in the year 2003.

Thioredoxin and glutaredoxin are considered to be the reducing systems in placenta. One of the recent studies had analysed this system present in the placenta of women with preeclampsia. In this study, they have analysed the tissues from placenta of normal pregnant ladies and also in females with mild to severe preeclampsia. The samples have been collected as early as possible after the delivery. And it was found that the above mentioned proteins have been elevated nearly 3-4 times in the placenta of preeclampsia women in comparison with that of placenta from normal gestation. This indicates that the placentas of preeclampsia women have been continuously under oxidative stress. It also shows the adaptive induction of thiol/disulphide oxidoreductases in cases of

preeclampsia.This was proven in the study done by Pankaj in the year 2008. .

Other related observations pointing placental factor as the cause of preeclampsia.

1 The studies done by Fisher et. al in the year 1999, study of August et.al in the year 1995 as well as that done by Hanretty et al in the year 1988.

Observe that placental hypoxia as the cause for pathogenesis of preeclampsia.

In the early stage of gestation, spiral arteries present in the uterine musculature will be invaded by the tissues of cytotrophoblast. This will result in the replacement of endothelial lining present in those vasculature. By end of second trimester there wont be any endothelial lining for spiral arteries and it is replaced by cytotrophoblast.

This change of the spiral vessels present in the uterine musculature leads to decrease in resistance offered by arterioles. This is accompanied by an enhanced blood flow to the foetus. The cytotrophoblast, in cases of preeclampsia, will be invading only up to proximal portion of decidua and nearly 40%-50% of spiral vessels present in the placental system will escape from the trophoblast induced remodelling of vascular system. This

is from the study done by Roberts et al in the year 1991 and Brosens et al in the year 1972.

Other important observation for the significant prominence played by placenta as a causative factor of preeclampsia is the sudden improvement & recovery of the condition after the delivery has taken place.

Immunological Factor

CD3 Zeta Protein

Pre-eclampsia is featured by reduced inhibition of expression of CD3-zeta protein ,leading to altered functionig of T-cells when a comparison has been made with normotensive pregnancies. CD3-zeta has been found iside the membranous area of the receptor for T cells which is neede for the multiplcation of T cells.

T cell mediated immunity is suppressed in normal pregnancy. Normal gestation has been shown to have low expression of CD3-zeta protein which is required to suppress cell mediated immunity inorder to save the fetus from rejection of allograft.

All these studies observe that there may be some factors that down regulate CD3-zeta, and pre eclampsic patients are lacking this, leading to increased inflammatory response.

Sleeping Disorders Breathing in Pre-eclampsia

Sleep-disordered breathing is divided in to snoring, upper airway resistance disorder & also in to obstructive sleep apnea-hypopnea disorder. These constitute snoring at one end and OSA at the other end. These are associated with oxygen desaturation or sleep fragmentation. Estrogen causes hyperemia and nasopharyngeal edema which can lead to upper airway obstruction. This is from the study of Young et al in the year 2001. According to the work done by Young et al. in the year 1997 it has been observed that this sleep disordered breathing is more common in patients with preeclampsia.

Significance of the renin-angiotensin mechanism in the pathophysiology of pre-eclampsia AbdAlla et al., (2001), Nielsen et al., (2000).

Pre-eclampsia has been considered as a disorder with elevated blood pressure that has been proven to be unique in having persistent involvement of the renal system. Renin-angiotensin system (RAS) is the system which is postulated in the etiopathogenesis involved in preeclampsia. This model has made the observation that a RAS which is tissue-based has been found in uteroplacental system. Stimulation of uteroplacental tissue RAS leads to the entry of angiotensin II in to

systemic vasculature and this might contribute to the mechanism involved in pre-eclampsia.

Other observation with RAS

1 Various components of this system including renin, angiotensinogen I& II, as well as aldosterone have been elevated in normal pregnancy Skinner (1972)

- increased adrenal sensitivity to ANG II Symonds et al., (1972) in p preeclampsia
- incresed level of ANG II for a given level of renin in preeclampsia
- Association of preeclampsia with enhanced sensitivity to angiotensinogen II Gant et al., (1973)

Role of Adenosine Receptor Expression. [Hasko and Cronstein 2004], Ajamieh et al.,(2008)

Adenosine is derived from the metabolism of adenine nucleotides. It has been secreted in various cells including placental tissue, as a reactive mechanism to decreased oxygen supply, decreased blood supply & also to inflammation. It function as a regulator of vascular resistance, angiogenesis, inflammation & oxidation induced stress. This is based on

the study done by Ajamieh et al in the year 2008.Particular receptors of adenosine are present which mediates the various physiological effects.

According to the studies done by Yoneyama et al in the year 2002., it has been shown that the concentration & upregulation of adenosine receptor tends to be substantially increased in placenta of females with preeclampsia

FUNCTION OF VASCULAR ENDOTHELIUM [Taylor et al, (1999) and Roberts's et al, (1989)]

Taylor et al in the year 1999 & Roberts's et al in the year 1989. have studied about the vascular endothelial functions.

The functions of vascular endothelium include

- the regulation of tone of smooth muscle
- secretion of various factors that could result in constriction as well as dilatation of blood vessels
- through secreting various soluble agents it regulates not only anticoagulation & antiplatelet function, but also fibrinolysis.

Placental secretion of these substances as a result of decreased blood supply will cause abnormalities in functions of endothelium.

The observation that the defect in the function of endothelium as initial mechanism of preeclampsia, suggests this as a probable cause for pathogenesis of PE.

ENDOTHELIAL DYSFUNCTION

Markers of endothelial dysfunction

Plasma thrombomodulin.

Von Willebrand factor

Fibronectin

TNF alpha

IL-1 levels

IL-2

Preeclampsia is a disorder of cells of endothelium. Several indicators of abnormalities of functions of endothelium have been found elevated in serum of patients with preeclampsia. This has been shown in the studies done by Taylor et al in the year 1999 & Roberts et. al in the year 1989.All these markers were studied in preeclampsic patients by various investigators .the majorities of these studies reported a significant association with this biomarkers. Sunder et al., (1989) Vince et al., (1995).

Elevated assay of platelet endothelial adhesion molecule-1 is shown to be associated with PE. This has been observed in the works of

Taylor et al & Roberts's et al. From these studies it appear that platelets play an important role in the pathogenesis of pre-eclampsia.

One among the other vascular adhesion molecules that are found elevated in preeclampsia is vascular adhesion molecule-1. In addition to this intercellular adhesion molecule-1 is elevated along with an elevation in E-selectin level.

Risk Factors of Developing Preeclampsia Adelusi et al., (1986), Steegers et al., (1998)., Visser et al. (1999). Magnussen et al. (2007)

Females with an incidence of pre-eclampsia in their previous gestations have an increased rate of developing the disease in the subsequent pregnancies. This is shown in the study of Adelusi et al in the year 1986.According to study done by Steegers in 1998,there is an incresed risk with advancing age of gestation. Based on the severity of protein excretion in urine, the risk of developing preeclampsia in subsequent gestations could be assessed.

Degree of protenuria in their previous pregnacy was studied by Visser et al. (1999) and has got leanier relationship with development of pre-eclampsia in future pregnancies.

Blood pressure in previous pregnancy tends to show a positive relation with development of preeclampsia. This has been suggested in the work of Magnussen et al in 2007.

Female sex hormone

As per the study done by Patrizia et al in 1999, these hormones have a profound influence on serum lipid profile. Towards later part of gestation, estrogen lowers the secretion of VLDLs and inhibit lipolytic activity taking place in mother. This is based on the study of Karl et al in 2003.This is a protective mechanisam so that more triglycerides will be shunted towads fetoplacental system inorder to cope up with the increased demand of fetus for the nutrition. The role of estrogen in increasing density VLDL formation & lowering the action of liver lipase has a significant value in pathogenesis involved in PE. (Based on the study of Alvarez et. al, in the year 1996).

FUNCTIONS OF ESTROGEN

1. Enhances the dilation of blood vessels
2. Suppress the reaction of vascular system to injury process.
3. Prevent the occurence of atherosclerosis.
4. Increases the bioavailability of endothelial-derived nitric oxide.

Alterd Estrogen-induced vasodilatation can be an important pathogenetic mechaniasm for the development of PE. Register et al., (1998).

Nitric Oxide and pre-eclampsia

Significant data shows that Nitric oxide (NO) formation is increased in normal gestation. (Based on the study by Sladek et al in 1997). Increase in synthesis of NO acts as a key factor in the dilatation of vessels in the pregnant kidney. (according to the study by Sladek et al in 1997). NO function as a significant factor regulating vasodilation during normal gestation, so decreased NO production of preeclampsia may play a role in the disease process.

Homocysteine in preeclampsia

Homocysteine which is one of the essential amino acid is needed for normal growth of our cells. Its elevated levels in maternal serumhas been related to various disorders mediated by placenta like PE. (Based on the study of Mignini et al. in 2005). Its quantity has been considerably elevated once preeclampsia is established. (Ingec et al., 2005).Due to inadequacy of consistent records, this relationship that has been noticed could not be regarded as a causative factor according to present literature.

BMI & Preeclampsia [Sahu el al., (2007), Bhattacharya et al (2007), Leeners et al., (2006), Ramos et al., (2005), Kabiru et al., (2004).] Females who have been grouped as obese according to BMI values in the 1st 3months of gestation have been found to develop lowered

endothelium-dependent and independent dilation of vessels when comparison was drawn with lean women. (based on the study by Stewart et al., in 2007). Another study done by Ramsay et al in the year 2002, suggested that higher BMI in initial part of gestations has been related with elevated systolic BP, triacylglycerol level and bring down level of HDL.

Higher BMI in the initial phase of gestation tends to show frequent correlation with risk parameters of eclampsia (based on study by Sahu et al. in year 2007 & Bhattacharya et al in the year 2007.

Diet & Nutrition in Preeclampsia

The association between pre-eclampsia and inadequacy in nutrition both prior to conception as well as during conception had been observed in several studies. One such example was the suggestion of including calcium mineral & fish oil in the diet of pregnant mothers. This was based on the analysis from several studies which showed a relationship between food intake & prevalence of pre-eclampsia among several groups.

Studies done by Mahomed and Atallah et al showed that by adding zinc & magnesium in the diet have been helpful in maintaining the general physiological mechanism of pregnancy period. These studies

were carried out in the year 2008 and 2004 respectively. Research work done by Makrides, which was carried out in the year 2008, showed that introduction of various antioxidants including vitamin C & E, selenium & garlic in the diet can compensate for the oxidative stress .Study also stressed the role of folic acid in bringing down the elevated serum homocysteine values.

Several nutritional parameters have also been related to pre-eclampsia condition. Various studies have been done among the Mayan Indians in Guatemala, who showed the habit of soaking corn inside lime prior to cooking process. Because of this habit of the people in those regions, their diet was rich in calcium and the rate of pre-eclampsia as well as eclampsia was minimal among them. Based on this observation ,it was suggested that by enhancing the calcium content of pregnant diet it could be possible to achieve a reduction in the prevalence of pre-eclampsia in the females having less calcium .

The study done by Dyerber et al among the Greenland Inuits population who consume plenty of oily fishes showed that the prevalence of pre eclampsia in those subsets was minimal. This observation further emphasized the role of fish oil in the prevention of eclampsia.

According to this study, not only fish oil, but also minerals like magnesium, zinc, selenium & several vitamins that are proven to have

antioxidants properties like vitamin C & E as well as folic acid have a significant role in decreasing the incidence of pre eclampsia.

Adequate nutrition is essential for the normal growth of foetus. Hence, dietary factors play a prominent role in the health of pregnant females.

Studies done by Krammer which was in the year 2000 and Laurence et al in the year 1980 showed that inadequate nutrition among pregnant mothers had resulted not only in abortion and decreased growth, but also in learning defects & personality disorders in the children.

In this scenario, the role of antenatal health workers is very significant. They should be educated and trained properly so that they will provide iron, calcium & vitamin tablets to the pregnant mothers.It is very essential to have a good idea about the diet & nutrition of pregnant female. This will help in the implementation of nutrition and education programs effectively.

Studies done by Picciano (which was in the year 1996) had shown that there is an enhanced reqirement of energy as well as nutrients in the pregnant period, which inturn is dependent on the built and nourishment of those ladies. The need for extra energy is low for females with good nourishment as their body have a better adaptive mechanism. Show only a minimal

An average sized Indian female, in her final three months of antenatal period, should take 2000 kilo calories per day on an average. However, many ladies in developing nations reduce their food consumption in pregnancy inorder to get a small baby. This is based on their belief that as the size of the baby decreases, obstetric complications become lesser. This was found in the study done by Brems et al in the year 1988.

But the current datas prove that new borns with small birth size tends to show more medical problems in future life. This was stressed in the study done by Godfrey and Barker.

The greatest essential principle that needed to be followed by a pregnant mother is to take a diet which is balanced as well as healthy. Such a diet consists of food material from various categories in adequate quantity inorder to ascertain correct nutrition. She requires all essential nutrients in adequate proportion.

The role of nutrition in organ development, growth and reproductive process is very importantl. It also play a vital role in enhancing the body's immune status as well as in the prevention of various disease processes.

Pregnancy, though a normal physiological process, is perhaps a stressful situation to the human body. Hence, this is a condition which

demands enhanced intake of nutrients so that body's extra requirements are fulfilled. This was well pointed out in the study done by Amy, in the year 1995.

According to the study done in Harvard, prevention of pre-eclampsia was possible by consuming a minimum of seventy five grams of protein daily. When a female becomes pregnant, her blood volume gets elevated by nearly two fold .Hence a corresponding increase in her nutrient intake to meet the body's requirement is absolutely essential.

Management of Pre-eclampsia

Pevention of pre-eclampsia is infact a real challenge. The steps and measures taken for its prevention purpose have not been successful. (Sibai 1998).

A meta analysis of fourteen trials which was done by giving low dosage of aspirin (a dose of 60-150mg per day) in mothers with risk parameters for preeclampsia showed that the incidence of preeclampsia as well as perinatal mortality has been decreased with the usage of drug aspirin. But it has failed to show any impact on birth weight or any reduction in the rate of abruption. (based on the study done by Coomarasamy et al in the year 2003). There was only a minor reduction in the occurrence rate when aspirin in low dosage was given to unselected

nulliparous females. (From the study of Sibai & Caritis in the year 1993).But it is justifiable to administer low dosage aspirin to high risk mothers ,starting from the 12th week of pregnancy. The safety profile of using aspirin in small dosage in 2nd & 3rd trimesters has been clearly proven.

Antihypertensive Medications

The sudden rise in blood pressure can result in catastrophic cerebrovascular & cardiovascular events. These constitute the important reasons of mortality & morbidity among pregnant mothers. Hence, the timely and proper management of elevated blood pressure in pregnancy plays a significant role.

The antihypertensive therapy has been proved effective in the prevention the above mentioned adverse events. However, in mild preeclampsia it has not proven to be effective. This was observed in the study done by Bolte et al, that was done in 2001.

The latest evidences from Cochrane review showed that the role of blood pressure lowering medications in mild to moderate elevation of blood pressure in pregnant period is uncertain.

This was further stressed by the meta analysis done in blood pressure lowering medications in pregnancy. It had shown that the usage

of these medications in females with mild form of disease enhanced the possibility of having a small sized baby.

It has been adviced by few people that BP lowering drugs could be used if there is a persistent elevation of systolic blood pressure of minimum 160mmHg & diastolic level of minimum 110mmHg.

Intravenous hydralazine, labetalol & nifidipine which is of short duration of action taken orally have been the frequently used drugs for the treatment of sudden rise in blood pressure in pre-eclampsia females. Among the above, hydralazine given through IV route has been regarded as the medication of choice by various people.

A meta analysis of twenty one studies was done by Magee & his colleagues & it was done in 893 females .Of that 8studies were on the comparison between hydralazine and nifedipine & 5 studies were on the comparison of hydralazine & labetalol. The work found that drug hydralazine has been related with substantially greater adverse effects in mothers & had a very poor maternal as well as perinatal outcome when comparison was made with other two drugs. Analysis had also found the essentiality of properly monitored clinical trials for the comparison of labetalol & nifidipine in the therapy of severely elevated blood pressure among preeclampsia females. Infact among the various medications used

for lowering the blood pressure, it has not been finalised to consider which single medication as the drug of choice.

In the subgroup analysis which was carried out, it was suggested that the efficiency of beta blockers might be low when a comparison was made with blockers of calcium channel. This work was carried out by Papatsonis et al & also by and Tsatsaris et al in the year 2001.

Pattern of lifestyle

It was suggested that rest as well as exercise could influence blood pressure level. The role of variations of activity pattern of females when they become pregnant in the incidence of preeclampsia is not yet certain 2small trials that have been carried out among the high risk females with normal blood pressure, have shown that those who were taking rest for a duration of minimum 4hours during day time had decreased incidence of preeclampsia. However, among the hospitalised hypertensive females the role of rest is not very certain. Also it is not clear whether the habit of taking rest had any extra benefit over routine activity pattern.

Few studies were of the opinion that the increased level of exercise has the potential to bring down the incidence of preeclampsia. But none of these datas were proven to be statistically significant. Hence, the decision to take rest or to do more exercise should be based on the

particular individual's personal opinion. This was the suggestion from the study of Lelia et al in the year 2006.

Recurrence

The incidence of preeclampsia among females who had history of the same in their prior pregnancy was found to be nearly 10 percentage according to the study done by Chesley in the year 1978.Based on studies of several research workers, females with history of severe preeclampsia & eclampsia carried a 20perceentage risk of having the same in her next pregnancy..

Seizure Prophylaxis

The medication of choice for the prevention of epilepsy among preeclampsia mothers is magnesium sulphate. The exact mechanism by which magnesium sulphate prevents epilepsy is not clear. However, various randomised trials had made the observation that the above drug has extra benefits over other drugs like benzodiazepam, phenytoin in the prophylaxis of early eclamptic fits as well as recurring epilepsy. Study done by Sibai in the year 2004 also strengthened the above finding.

The treatment has to be initiated at the time of onset of labor or before the cesarian & it has to be given for next one day after the delivery.

According to the severity of the underlying disease, the decision to prolong the use of medication after delivery can be formulated. Therapy is initiated with a loading bolus of parentral magnesium sulphate, which is generally four to six grams & it is followed by maintenance dose which is usually one to three gram per hour.

Magnesium sulphate act by inhibiting calcium transport as well as the aggregation of platelets & also by dilating the cerebral vasculature. These are the mechanisms that has been suggested for the antiepileptic action of the drug. (According to the study done by Sibai in the year 2004).

But those individuals who are treated with the above medication requires intensive monitoring to identify the features of magnesium induced toxicity very early itself. Various manifestations of toxicity include absence of knee jerk, respiratory difficulty as well as decreased urine output. This emphazise the need of examining knee jerk atleast fourth hourly. Rate of respiration as well as saturation should also be kept under close observation. There should be urine output of minimum thirty millilitre per hour.

Serum magnesium value has to be found out if there is a doubt of toxicity

Majority of the obstetricians, prefer to keep the value less than 9mg/dl.But the features of toxicity has been noted even at a value less than 6mg/dl.Hence, irrespective of the serum level close monitoring of the individual has to be effectively carried out. This has been pointed out by Sibai in the year 2004.

Every female who is in labour with features of severe preeclampsia, has to be treated with magnesiumsulfate inorder to prevent epilepsy. But there is a controversy regarding the role of magnesium in the prevention of seizures in cases of mild preeclampsia. Though ACOG has adviced to use magnesium in severe cases of preeclampsia,it has not advocated the use of above drug in mild forms of preeclampsia.Certain obstetricians avoid the use of magnesium if the blood pressure has been controlled &/or only increased to a small extend & if the count of platelets is within the normal limits. However, some practioners suggest magnesium therapy even to those with PIH as a minor proportion of these women show a tendency to progress to preeclampsia. Study done by Sibai in the year 2004 showed that it is essential to give magnesium to approximately 100 women with PIH inorder to avoid one eclampsia case.

Plasma volume expansion

Certain females with severe form of preeclampsia tend to have elevated haematocrit & decreased plasma volume. Based on this

observation, the use of colloids or crystalloids for the expansion of plasma volume has been advocated inorder to enhance the maternal as well as uteroplacental blood flow. But the condition of volume overload has to be avoided as it can result in life threatening pulmonary or cerebral oedema. Moreover, higher rate of volume expansion should be done with the monitoring of central venous pressure which is an invasive method & these procedures by itself have a significant risk. This has been shown in the studies of Bolte et al in the year 2001 .

MATERIALS AND METHODS

MATERIALS AND METHODS

Materials

Source of Data

This study was conducted at Mahatma Gandhi Memorial Government Hospital, Trichy, Tamilnadu in colloboration with department of obstetrics and gynaecology during the period of january 2011 to august 2012.

Study Design

Single centre, cross-sectional and analytical study.

Period of Study

The work was carried out from january2011 to august, 2012.

Ethical Committee Approval

Ethical committee approval obtained from the Institutional Ethical committee.

Inclusion criteria

Study were conducted on 90 women ranging in age from 20 to 30 years. They were grouped in to three. All the pregnant participents were in their 3 rd trimester with single intrauterine pregnanacy.

GROUP A-preeclampsic women -after 20 wks of pregnancy with Blood Pressure ≥ 140/90 and proteinuria (positive dipstick).

GROUP B-normotensive pregnant women-individuals with Blood Pressure ≤120/80 for control.

GROUP C-normotensive non pregnant women- relatives of some patients and some staff workers of labour ward willingly consented to take part in this study.

All the participants were inquired by a questionnaire containing their personal history, family history of PIH, Twins, Hypertension and Diabetes, Drug history and their symptoms.

Exclusion criteria

Women with past history of diabetes mellitus, hypertension, kidney diseases, hepatic dysfunction and dyslipidemia were excluded from the study.

Consent

An informed consent was obtained from all the participants.

Methods of collection of data

Blood sample collection

Neraly 8ml of blood was collected in the fasting state & the below mentioned factors have been analysed in cases as well as in controls. Blood lipid parameters were estimated with the help of autoanalyser based on enzymtic colorimetric process. This include not only triglyceride (TG) & total value of cholesterol (TC), but also the value of HDLcholesterol (HDL-C). As per Friedewald's formula serum values of LDL & VLDL were found out. That is LDL Cholesterol = Total cholesterol - (triglyceide/5+ the value of HDL cholesterol) (TG/5 + HDL-C) The value of VLDL is Triglyceride/5

Blood pressure recording

BP was recorded in sitting position with shygmomanometer. BP was recorded after the subject had a rest of minimum ten minutes.Based on korotkoff sounds BP was recorded. BP at which the sounds started to appear had been taken as systolic value of BP and that when sound disappears had been taken as diastolic pressure. The size of the BP cuff used was 12x22cm .

Calculation of body mass index

Anthropometric measurements

Height was recorded with tape to the nearest one centimeter.

Subjects were instructed to stand upright without shoes with their back against the wall, foot together and eyes directed forward.

Weight was measured with weighing machine using spring balance that was kept on firm horizontal surface. The scale was checked on daily basis and calibration was done with known weights. Weight was recorded to the nearest 0.5 kg.

The formula given below has been used to calculate BMI

$$BMI=\text{Weight (Height In Meters)}^2$$

URINE ANALYSIS

Midstream urine of the subjects that has been collected in the early morning has been analysed. Females were informed about the method to collect urine in the specified bottles. With the help of dipstick method analysis of collected urine was carried out.

Analysis of data

The information collected regarding all the cases were tabulated in a master chart. Data was analysed with help of statistical soft ware tool **epidemiological information package (EPI 2002).** Using this software range, frequencies, percentages, means standard deviations, and p values were calculated. For comparison of multiple groups ANOVA test was used. Scheffe Post Hoc Test was used to find out the p value between the means. Pearson Correlation coefficients were used to find out the correlation between the variables.

RESULTS

RESULTS

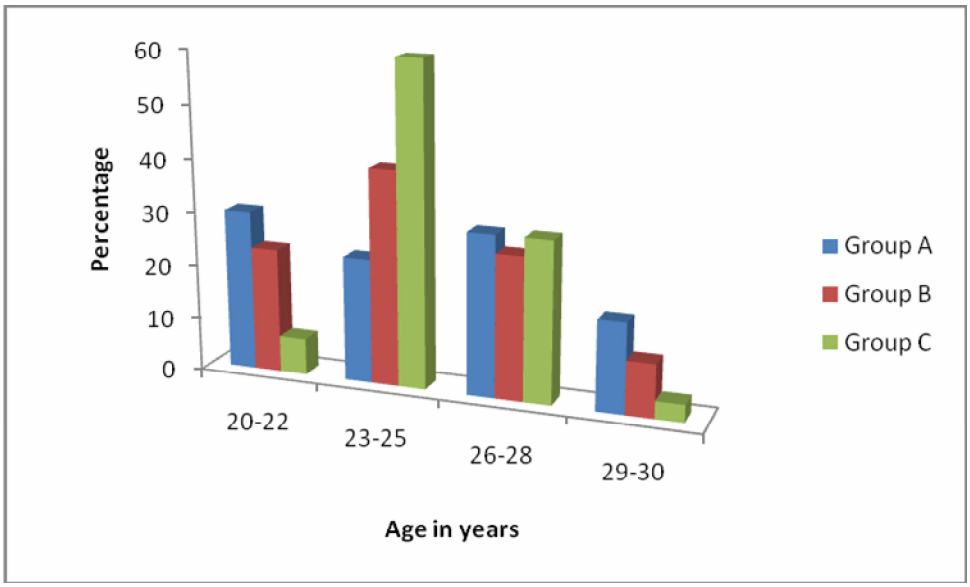
Table 1 A

Age distribution among the groups

Age	Group A		Group B		Group C	
	N	%	N	%	N	%
20-22	9	30.0	7	23.3	2	6.7
23-25	7	23.3	12	40.0	18	60.0
26-28	9	30.0	8	26.7	9	30.0
29-30	5	16.7	3	10.0	1	3.3
Total	30	100.0	30	100.0	30	100.0

Table 1B

	Age in years		
	Group A	Group B	Group C
Minimum	21	20	22
First quartile	22.0	22.8	23.8
Median	25.0	25.0	25.0
Third quartile	27.0	27.0	26.0
Maximum	30	30	30



BAR DIAGRAM SHOWING THE DISTRIBUTION OF AGE AMONG THE GROUPS.

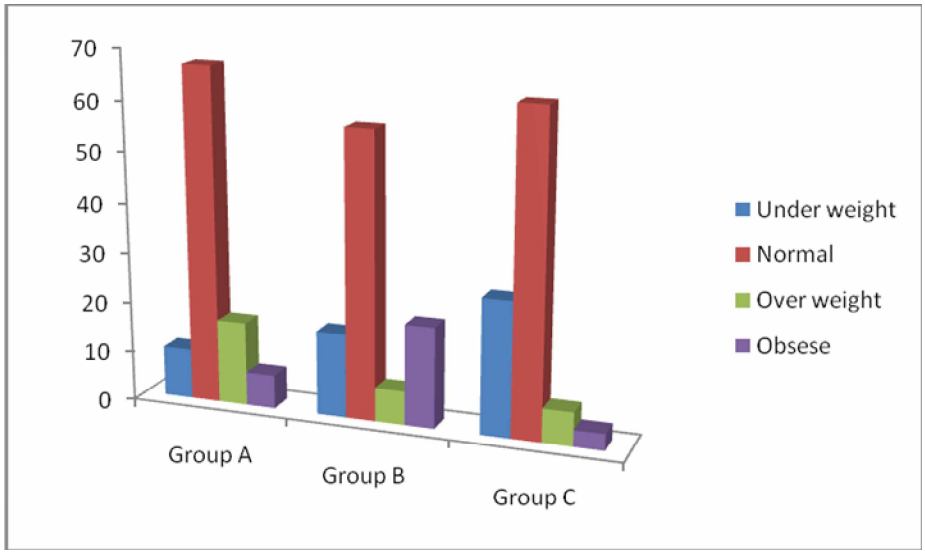
Result –age is distributed among the groups, hence they are comparable.

BMI

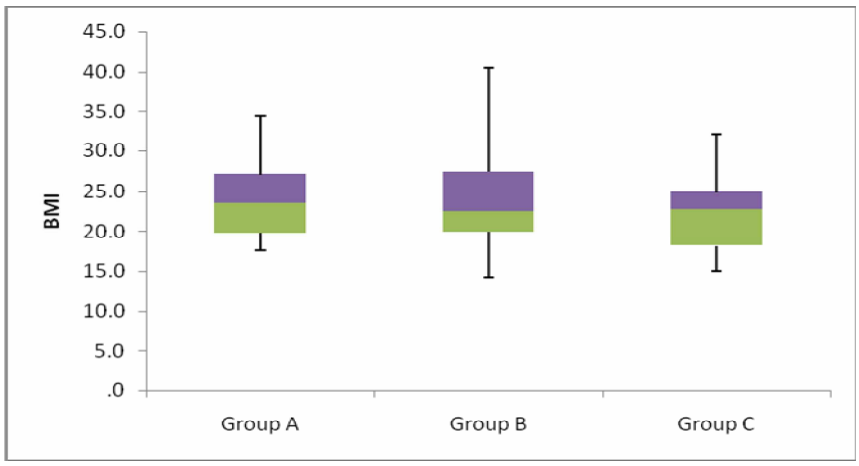
Table 2A

Distribution BMI among the groups.

BMI	Group A		Group B		Group C	
	N	%	N	%	N	%
Under weight	3	10.0	5	16.7	8	26.7
Normal	20	66.7	17	56.7	19	63.3
Over weight	5	16.7	2	6.7	2	6.7
Obsese	2	6.7	6	20.0	1	3.3
Total	30	100.0	30	100.0	30	100.0



BAR DIAGRAM SHOWING THE DISTRIBUTION OF BMI AMONG THE GROUPS



Box plot diagram describing BMI:- Lower and upper end of the whisker represents minimum and maximum BMI respectively. Lower border of the rectangular box represents the 25th percentile, upper border represents 75th percentile and the line of separation (middle line) of the two coloured box represents the median BMI.

Table distribution among the groups.

Comparison of BMI

	N	Mean	sd	F	p
Group A	30	23.49	4.49	.970	.383
Group B	30	23.91	5.98		
Group C	30	22.20	4.19		
Total	90	23.20	4.95		

Table

Post hoc test	Multiple comparison		p
BMI	Group A	Group B	.748
	Group A	Group C	.314
	Group B	Group C	.185

RESULT

There is no significant difference in BMI among the groups, hence they are comparable.

TOTAL CHOLESTEROL

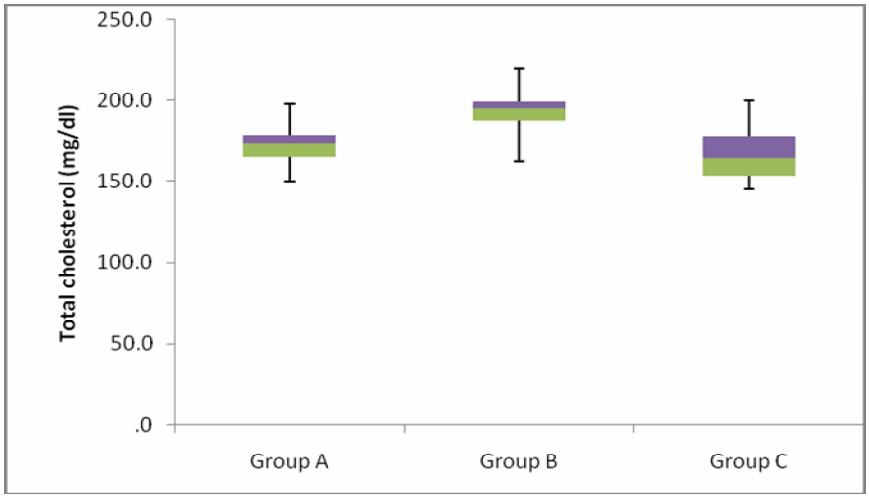
Table3A

Comparison of Total cholesterol in (mg/dl)

Normal Value: <200mg/dl

	N	Mean	sd	F	p
Group A	30	172.5	12.1	39.032	<0.001
Group B	30	192.2	11.2		
Group C	30	165.2	13.4		
Total	90	176.6	16.7		

Box plot diagram describing distribution of Total cholesterol



Post hoc test	Multiple comparison		P value
T.CHOL	Group A	Group B	<0.001
	Group A	Group C	.024
	Group B	Group C	<0.001

Result –group B showed high level of total cholesterol. P value is significant between A and B, and B and C.P value is not significant between group A and C.

TRIGLYCERIDE

Table 4A

Distribution of TGL among the groups.

Normal value <150mg/dl.

	in mg/dl		
	Group A	Group B	Group C
Minimum	169.0	100.0	100.0
First quartile	195.0	106.0	102.8
Median	204.5	110.0	109.0
Third quartile	252.5	118.5	122.3
Maximum	290.0	166.0	146.0

Box plot diagram describing distribution of Triglyceride

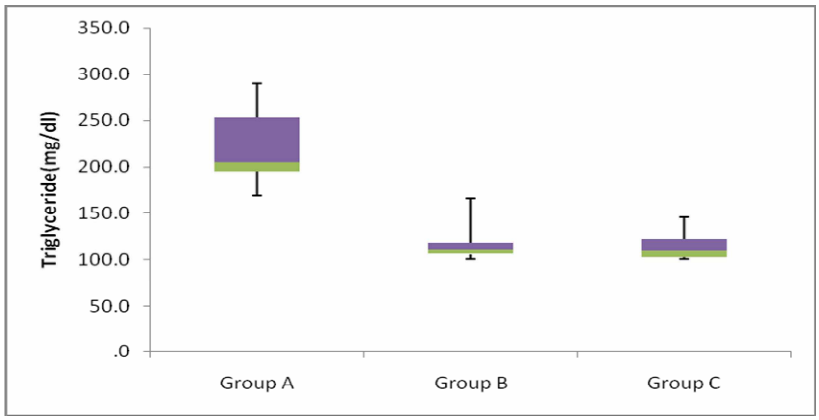


Table 4B

Comparison of Triglyceride in (mg/dl)

	N	Mean	sd	F	p
Group A	30	222.0	36.6	212.476	<0.001
Group B	30	113.8	12.1		
Group C	30	113.7	13.1		
Total	90	149.8	56.3		

Post hoc test	Multiple comparison		p
Triglyceride	Group A	Group B	<0.001
	Group A	Group C	<0.001
	Group B	Group C	0.983

Result - P value is significant between A and B ,and A and C.P value is not significant between group B and C.

HDL

Table5A

Distribution of HDL among the groups.

Normal value - >40 mg/dl

	N	Mean	sd	F	p
Group A	30	41.9	4.3	35.993	<0.001
Group B	30	46.9	3.0		
Group C	30	50.9	4.9		
Total	90	46.6	5.5		

Box plot diagram describing distribution of HDL

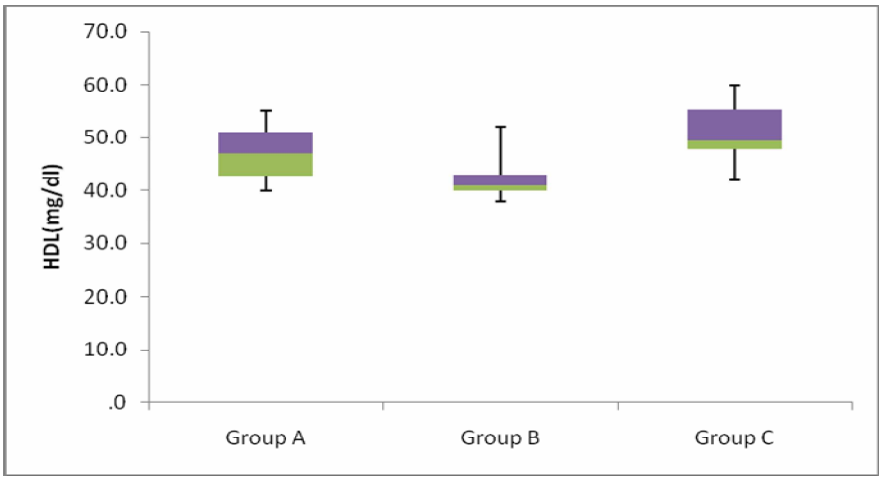


Table5 B

Comparison of HDL in (mg/dl)

Post hoc test	Multiple comparison		p
HDL	Group A	Group B	<0.001
	Group A	Group C	<0.001
	Group B	Group C	0.120

Result - P value is significant between A &B ,and A & C .

LDL

Table 6A

Distribution LDL among the groups.

Normal Value <130 mg/dl.

	N	Mean	sd	F	p
Group A	30	81.1	8.8	124.292	<0.001
Group B	30	127.6	11.4		
Group C	30	91.5	14.9		
Total	90	100.1	23.2		

Box plot diagram describing LDL

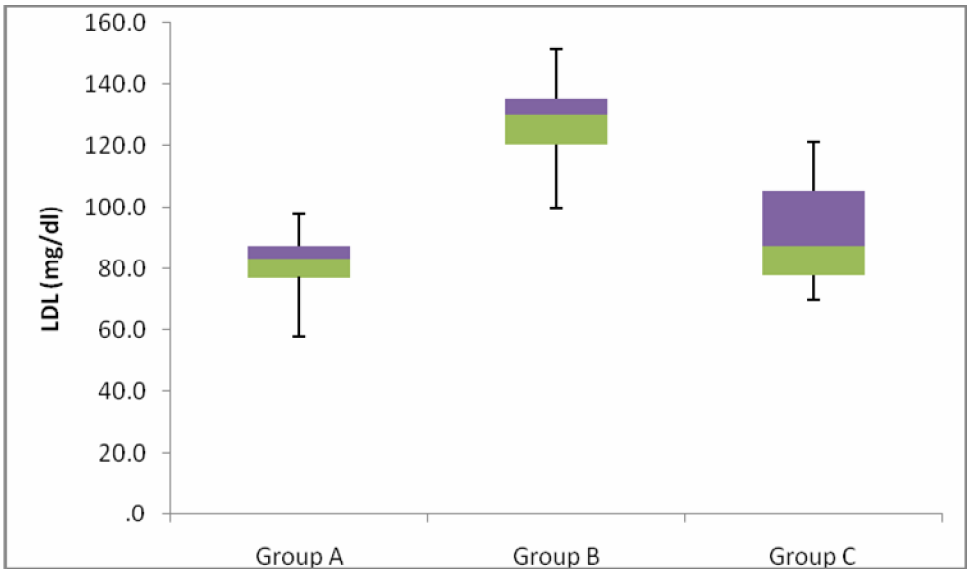


Table 6B

Comparison of LDL in (mg/dl)

Post hoc test	Multiple comparison		p
LDL	Group A	Group B	<0.001
	Group A	Group C	.001
	Group B	Group C	<0.001

Result - P value is significant between A & B and B & C.P value is not significant between group A and C.

VLDL

Table7A

Distribution of VLDL among the groups.

Normal value 10 – 30mg/dl.

	N	Mean	sd	F	p
Group A	30	44.4	7.3	212.476	<0.001
Group B	30	22.8	2.4		
Group C	30	22.7	2.6		
Total	90	30.0	11.3		

Box plot diagram describing distribution of VLDL

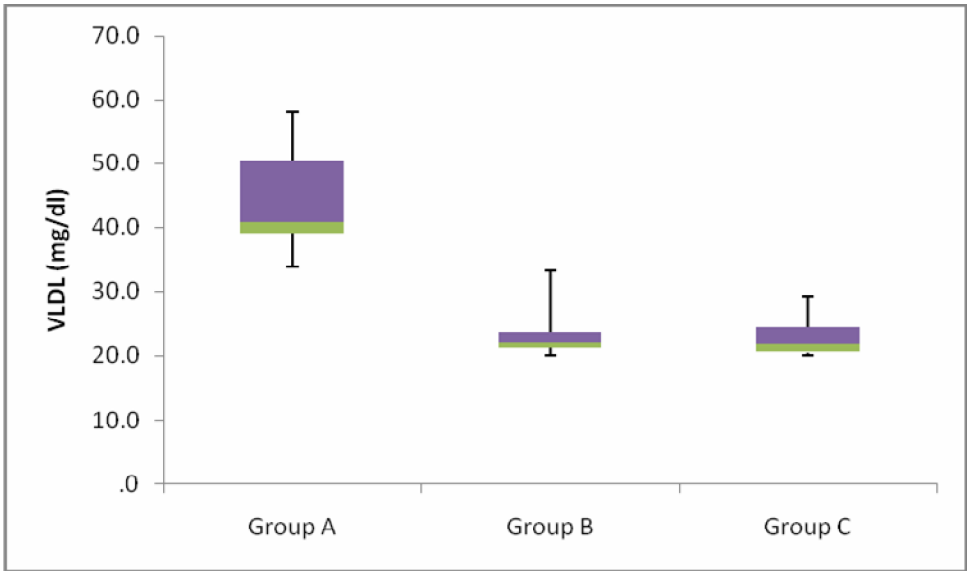


Table 7B

Comparison of VLDL in (mg/dl)

Post hoc test	Multiple comparison		p
VLDL	Group A	Group B	<0.001
	Group A	Group C	<0.001
	Group B	Group C	.983

Result - P value is significant between A & B and A & C.P value is not significant between group B and C.

BLOOD UREA

Table8A

Distribution of blood urea level among the groups.

	N	Mean	sd	F	p
Group A	30	24.7	5.7	5.113	.008
Group B	30	21.8	3.4		
Group C	30	21.5	3.1		
Total	90	22.7	4.4		

Box plot diagram describing blood urea level among the groups

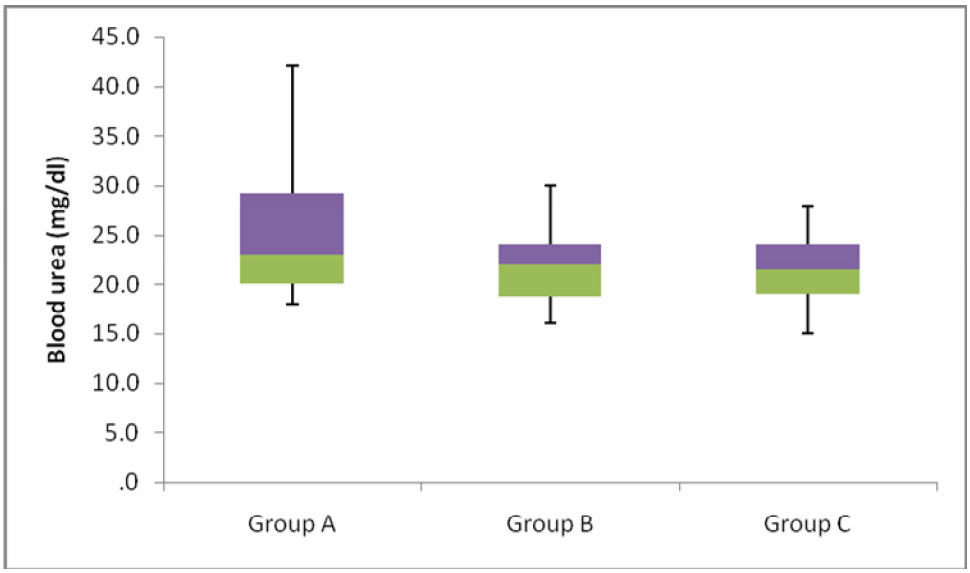


Table 8B

Comparison of Blood urea in (mg/dl)

Post hoc test	Multiple comparison		p
Blood urea	Group A	Group B	.010
	Group A	Group C	.005
	Group B	Group C	.784

Result –p value is not significant.

SERUM CREATININE

Table9A

Distribution of Serum Creatinine among the groups.

	N	Mean	sd	F	p
Group A	30	.81	.21	1.974	.145
Group B	30	.81	.16		
Group C	30	.73	.16		
Total	90	.79	.18		

Box plot diagram describing distribution of Serum Creatinine .

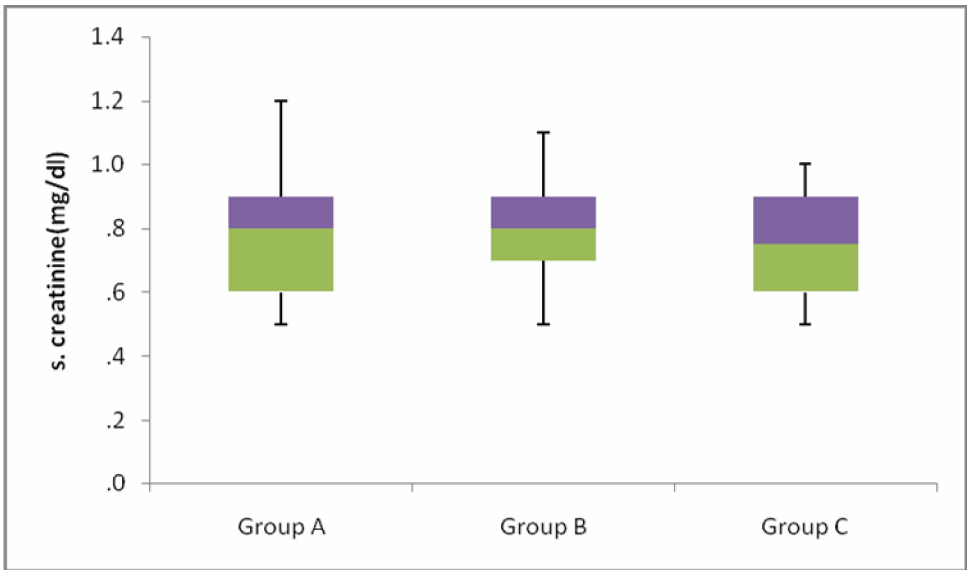


Table 9B

Comparison of S. Creatinine in (mg/dl)

Post hoc test	Multiple comparison		p
S. Creatinine	Group A	Group B	.942
	Group A	Group C	.083
	Group B	Group C	.096

Result –p value is not significant.

RISK RATIOS

TG / HDL

Table 10 A

Distribution TG / HDL among the groups

	N	Mean	sd	F	p
Group A	30	4.74	.69	209.201	<0.001
Group B	30	2.73	.35		
Group C	30	2.26	.37		
Total	90	3.24	1.19		

Box plot diagram describing

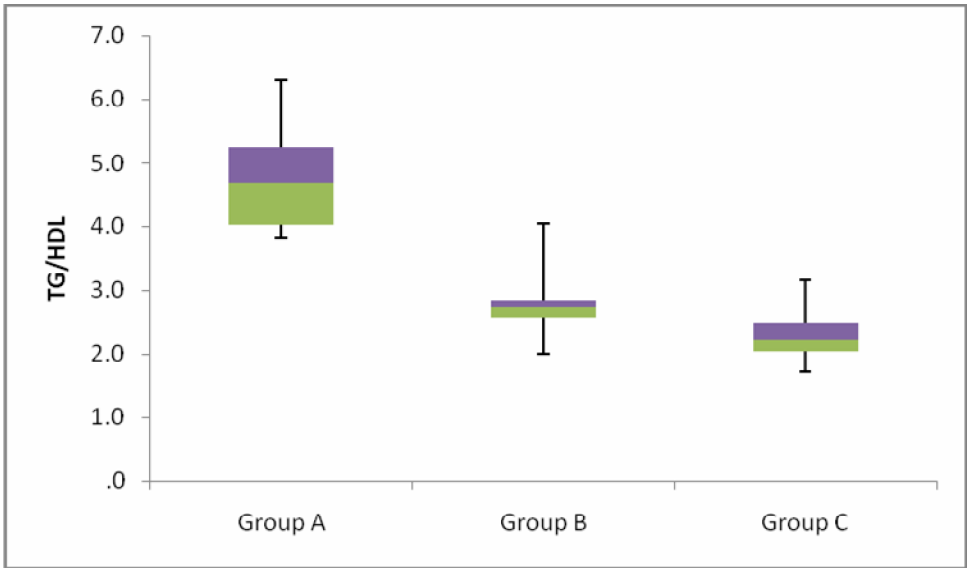


Table 10B

Comparison of TG/HDL

Post hoc test	Multiple comparison		p
TG/HDL	Group A	Group B	<0.001
	Group A	Group C	<0.001
	Group B	Group C	<0.001

LDL / HDL

Table11 A

Distribution of LDL / HDL among the groups.

	N	Mean	sd	F	p
Group A	30	1.75	.29	127.004	<0.001
Group B	30	3.06	.36		
Group C	30	1.83	.41		
Total	90	2.21	.70		

Box plot diagram describing LDL/HDL

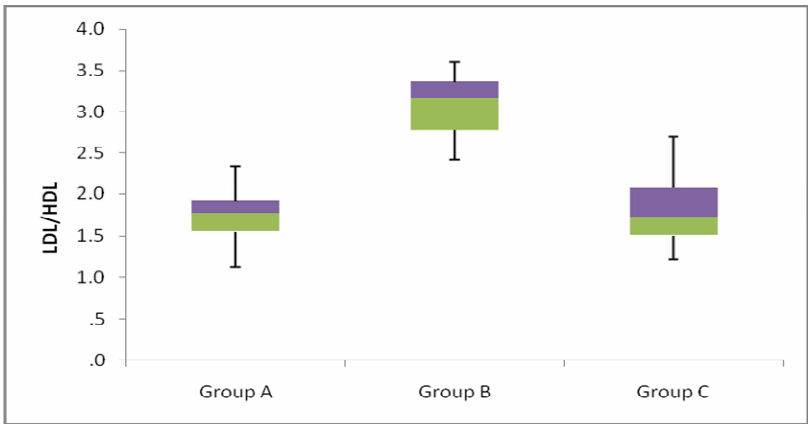


Table 11 B

Comparison of LDL/HDL

Post hoc test	Multiple comparison		p
LDL/HDL	Group A	Group B	<0.001
	Group A	Group C	.411
	Group B	Group C	<0.001

Result –p value is significant between group A &B, and group B&C.

HDL / VLDL

Table12A

Distribution of HDL / VLDL among the groups.

	N	Mean	sd	F	p
Group A	30	1.08	.15	162.418	<0.001
Group B	30	1.86	.23		
Group C	30	2.27	.36		
Total	90	1.74	.56		

Box plot diagram describing

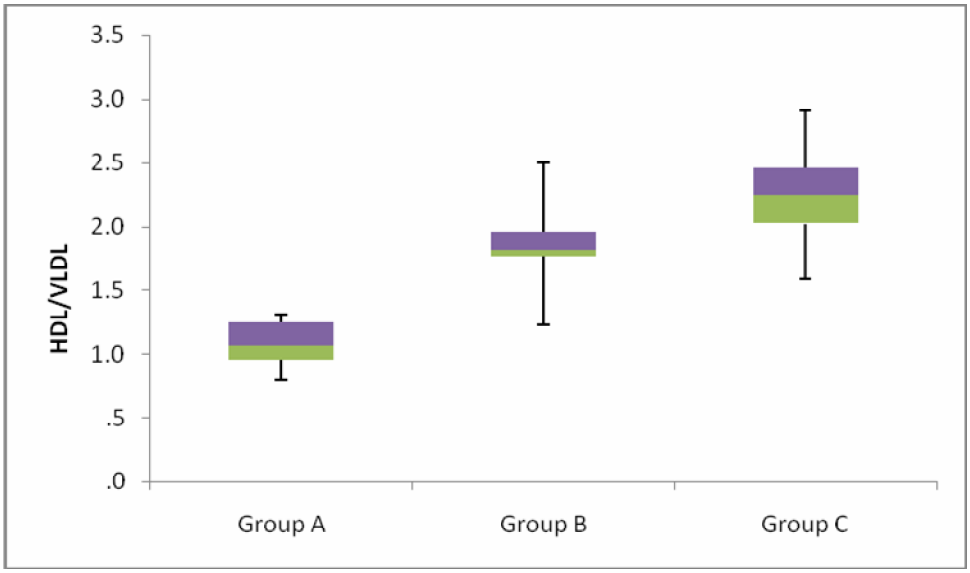


Table 12B

Comparison of HDL/VLDL

Post hoc test	Multiple comparison		p
HDL/VLDL	Group A	Group B	<0.001
	Group A	Group C	<0.001
	Group B	Group C	<0.001

Result – p value is very significant among the groups.

TC/HDL

Table 13A

Distribution of TC/HDL among the groups.

	N	Mean	sd	F	p
Group A	30	3.70	.35	86.958	<0.001
Group B	30	4.61	.39		
Group C	30	3.28	.46		
Total	90	3.86	.68		

Box plot diagram describing TC/HDL.

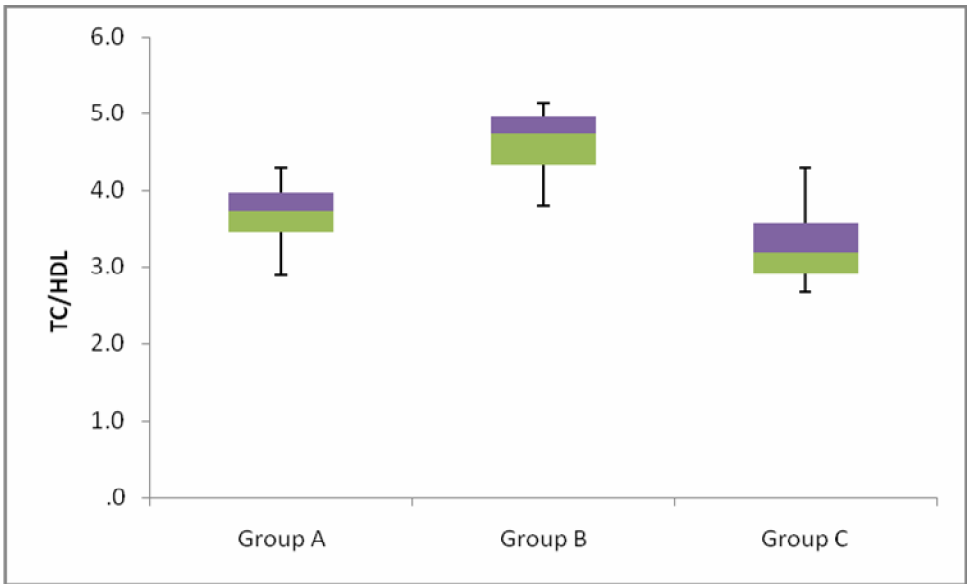


Table 13B

Comparison of TC/HDL

Post hoc test	Multiple comparison		p
TC/HDL	Group A	Group B	<0.001
	Group A	Group C	<0.001
	Group B	Group C	<0.001

Result p value is significant.

Correlation between Triglyceride and blood pressure

Table 14

Correlation between Triglyceride	**SYS.BP**	**DIA.BP**
Pearson Correlation	0.822**	0.865**
p	.000	.000
N	90	90

Result – There is highly significant positive correlation between TGL level with systolic and diastolic BP.

DISCUSSION

DISCUSSION

This study was conducted to determine the lipid profile changes in Preeclampsia and to compare this with normotensive pregnant and non pregnant women. The study population was 90 patients with 30 patients in each group. All the women involved in the study were between 20-30 years. It was ensured that none of the control group had diabetes mellitus, hypertension, dyslipidemia, renal, liver or any metabolic disorder. It was also ensured that none of the patients in the study group had any other medical disease. Serum total cholesterol, HDL cholesterol, LDL cholesterol, Triglycerides, LDL / HDL, HDL / VLDL, TC/HDL TG / HDL ratio were measured using an autoanalyser, after a 12 hour fasting. The results were statistically analysed.

Age distribution

The mean age for all the three group were 25 yrs. There was no significant difference in distribution of age among the groups.

Distribution of BMI among the groups

Among PE women 20(66%) were normal weight, 3 [10%] were under weight, 5[16%] were overweight and 2[6%] were obese.

Among normotensive pregnant women 17(56%) were normal

weight, 5[16%] were under weight, 2[6%]were overweight and 6[20%] were obese.

Among normotensive non pregnant women 19(63%) were normal weight, 8 [26%] were under weight, 2[6%] were overweight and 1[3%] were obese. The median BMI for PE women were 23.49 SD 4.49, for normotensive pregnant women were 23.91 SD 5.98 and normotensive non pregnant women were 22.20 SD 4.19.

We compared PE groups with normal pregnant women, the p value is not significant.[p value 0.748] . In some recent studies observed a high bmi **[**Sahu el al., (2007), Bhattacharya et al (2007), Leeners et al., (2006)] among preeclampsic women. In our study we didn't find any significant difference in BMI between preeclampsic and normal pregnant women.

Total cholesterol

Total cholesterol were normal for all the women with a mean 172.5 SD 12.1 for preeclamspsic group, 192.2 SD 11.2 for, for normotensive pregnant women and 165.2 SD 16.7 for normotensive non pregnant women. On statistical analysis we found that there is no significant alteration in cholesterol between normotensive non pregnant women and preeclamspsic group. But in our study total cholesterol was significantly elevated in normotensive pregnant women compared to both the other

groups [p value<0.001]. Thus we found that there is no significant change in TC in PE women and the values were less when compared to normotensive non pregnant women. These observations are similar to Sattar et al [10]. However some other studies observed that there is significant elevation in TC in women developing PE. Hubel et al, Adegoke et al.[11].

TGL

We observed that TGL were significantly elevated in PE women with a mean 222 SD 36.6 .For normotensive pregnant women mean was 113.8 with SD of 12.1 and for normotensive non pregnant women mean was 113.7 with a SD of 13.1. This is one of the important finding in our study. On statistical analysis this was very significant with p value <0.001.most of the previous studies[12] (Enquobahrie et al, Cekmen et al, Mikhail et al) observed this association and postulated that hypertriglyceridemia is an important factor in the pathogenesis of PE. We had a similar observation in our study.

HDL

In our study the mean HDL of PE women was very low (41.9 SD 4.3) compared to both the other groups. This was found very significant on statistical analysis with a p value of <0.001. Normal pregnancy is

associate dwith an incresse in HDL ,but in our study we observed that HDL values were at a lower level for normotensive pregnant women when compared to normal women.but this difference was not stastically significant [p value 0.120] . (6,7,9). This observations are consistent with previous studies[12]) (Bradley et al, Enquobahrie et al, Cekmen et al, Mikhail et al). This low level of HDL is due to both hypoestrogenemia and insulin resistance

VLDL

We observed that, there is no significant rise in serum VLDL level in normotensive pregnant women compared to non pregnant women,[p value 0.983]. This finding is against the observations made by some research workers.(Knopp etal, Teichmann et al) . However the mean value of VLDL in PE group were very high with a mean value of 44.4 sd of 7.3. This was statistically very significant compared with both the groups, p value <0.001. This is another important observation in our study. This observations is very much similar to studies done by (Sattar et al, Kokia et al, Teichman et al). This VLDL may cause injury to the vascular endothelium as evident from observations of Arbogast et al.

LDL

In our study, LDL was high in normal pregnant women compared to LDL in non pregnant women. [p value <0.001] .But for both the groups the level was under desirable limit (130 mg/dl) except for two women. We didn't find any statistically significant difference in LDL level between PE women with both the other groups. Even though our PE women showed a significant increase in TGL and VLDL level there was no much increase in LDL level, this is similar to the observation of. Wakatsuki et al.

TGL/ HDL RATIO.

We have also calculated the ratio of TGL to HDL, which is thought to be an important predictor of the outcome of PE .in our study we observed that the mean TGL/HDL ratio in PE group is 4.74 with SD of 0.69. In normal pregnant women the mean of the ratio was 2.73 SD 0.35 and for normal women it was 2.26 SD 0.37. On comparison this is statistically very significant [p value<0.001]. This observation is similar to the ongoing study Copenhagen Male Study (Jorgen Jeppesen et al).

HDL/VLDL

We have also observed that there is a significant fall in ratio of HDL to VLDL in PE group. This is statistically very significant when

compared with normal pregnant women. (p value <0.001). Similar observations were made by so many researchers. Enquobahrie et al and Kokia et al.

LDL/HDL RATIO AND TOTAL CHL/HDL

We have also calculated LDL/HDL RATIO and CHL/HDL ratio. When compared with normal pregnant women, PE group showed a less value with statistically significant p value [<0.001]. This one another observation which is against previous studies, which showed a high value of this ratio in PE consistently. [Enquobahrie et al and kokia et al]

This variation between the ratio can be explained by the normal value of HDL and total CHL in our study. We need further large scale study to establish the importance of this ratios in preeclampsia.

Relationship between blood pressure and trglycerides

In our study we also tried to find out the correlation of TGL level with blood pressure. We have observed that there is a positive correlation between TGL with systolic BP [r=0.822] and diastolic BP [r=0.865]. This is consistent with observations of Karl W et al 2003.

Pre-eclampsia a vascular disorder of pregnancy is one of the leading cause of maternal morbidity and mortality. Complications of hypertension are the 3rd important reason for mortality occuring in a

pregnant woman apart from that due to postpartum hemorrhage & embolism[13]. Disturbed lipid metabolism, was suggested as one of the mechanism for the pathogenesis of PE. In our study we found that all the lipid sub fractions were significantly elevated, more in the case of triglycerides. The significant rise in serum triglyceride concentration in pre-eclampsia in our study was established in the studies of many workers [14].

This elevation in TGL leads to increase in atherogenic small dense LDL concentrations. Activity of liver lipase was found as elevated in females with preeclampsia. This might lead to enhanced small dense low density lipoprotein concentration due to increased TGL exchange into LDL[15]. Value of HDL has been observed to be reduced, which might be perhaps due to elevated TGL values and elevated activity of liver lipase.[16].

One of the main reasons for this lipid dearrangement is due to the hormonal imbalance in preeclampsia. Preeclampsia is a state of hypoestrogenemia[17]. Reduction in utero-placental blood flow is the main pathophysiological event in preeclampsia which leads to inappropriate production of Dehydroepiandrosterone sulphate (DHEA) by fetal adrenal glands by interfering with the uptake and metabolisamof lipids by the fetus. DHEA is the most important source of estrogen in pregnancy. 90 %

of estrogen in a pregnant women is from fetal DHEA. Desulphuration and aromatization of DHEA by the placenatal enzymes sulfatase and aromatase forms estriol.[7] Because of this impairment in the formation of DHEA levels of estrogen in maternal blood decrease[18]. Another mechanisam for hypoestrogenemia is decrease in utero-placental blood flow causes stasis of blood in the intervillous space of placenta resulting in a decreased entry of steroids in to the maternal circulation causing hypoestrogenemia[19].

This state of hypoestrogenemia causes a decresed expression of VLDL / apo E receptors in the placenta which are essential for the lipid transport to the fetus. This results in reduced transport of VLDL to fetal side, which could be the reason for maternal hypertriglyceridemia. LDL which has to be taken up by the growing fetus for synthesising DHEA is decreased due to reduced perfusion fetoplacental unit leading to increased LDL. Thus a decreased catabolism of TGL by the placenta due to reduced blood supply and the simultaneous fall in the lipolysis of lipoprotein would contribute to the elevated level of triglycerides[20].

In a normal gestation, the formation of VLDL will be enhanced by estrogen. Also it plays a role in reducing the lipolysis mechanism. But due to the incresed VLDL/apoE receptor's expression which occurs in placenta, the lipoprotein are shunted to fetal circulation, meeting the

nutritional need of the growing fetus. Thus this elevation in triglyceride rich lipoprotein is physiologicaly very much imporatant for the fetus. But in pre-eclampsia the due to the dearrangement of placental blood supply TG rich lipoproteins are not cleared from circulation, exposing the vascular endothelium to the toxic effect. Both increased blood pressure and proteinuria might signify abnormal function of endothelium. Based on the study done by Argobast et al. It has been postulated that lipoproteins that are rich in triglycerides would cause endothelial damage in females with PE[(21)].

Blood Urea

We didn't find any significant difference in level of urea in blood between the groups .Mean value of urea for PE group was 24.7 SD 5.7, for normal pregnancy 21.8 SD 3.4 and for normal women it was 21.5 SD 3.1.This observations are consistent with previous studies [Lorencen et al].

Serum creatinine

Many of the old studies suggested that s.creatinine is increased in women with preeclampsia .However in our study there is no significant difference found with S.creatinine.

CONCLUSION

CONCLUSIONS

1. There is no significant difference in BMI between preeclampsic women and normotensive pregnant women.
2. There is no significant change in TC in PE women and the values were less when compared to normotensive non pregnant women.
3. Preeclampsic women showed high level of TGL which is very significant (p value < 0.001) compared to both normotensive pregnant and nonpregnat women.
4. Preeclampsic women showed low levels of HDL compared to normotensive pregnant women (p value < 0.001).
5. Preeclampsic women showed high VLDL level, which was very significant (p value < 0.001) compared to both normotensive pregnant and nonpregnat women.
6. There is no significant alteration in LDL level in PE women.
7. TGL/HDL ratio was very high in Preeclampsic women, which was statistically significant. (p value < 0.001).
8. HDL/VLDL ratio was low in Preeclampsic women which was statistically significant. (p value < 0.001).
9. TCL/HDL ratio and LDL/HDL ratio were low when compared to both normotensive pregnant and nonpregnat women.

10. We found a highly significant positive correlation between TGL level and systolic BP(r=0.822) and diastolic BP(r=0.865).
11. With this background we recommend that fasting lipid profile and risk ratio should be made as a part of routine antenatal checkup.
12. We recommend further large scale population study to establish the importance of risk ratios and TGL as a marker of preeclampsia.

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BIBLIOGRAPHY

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APPENDIX

PROFORMA

A COMPARATIVE STUDY OF LIPID PROFILE IN PREECLAMPSIA AND NORMOTENSIVE PREGNANT AND NONPREGNANT WOMEN.

1. Name : 2. Age

3. IP/OP NO:

4. Address 5. Occupation

6.

G	P	L	A

7.

N	N. PRE	PIH

8.Past history

CAD	HTN	DM	OTHER

9. Family history

CAD	HTN	DM	OTHER

10. General examination

R	B.P	R.R	TEMP	Wt	Ht	BMI

11. Systemic examination

CVS	R.S	ABDOMEN	C.N.S

12. Investigations CBC

HB	TC	DC	ESR	PCV	PLT

URINE ROUTINE

PROTEIN	SUGAR	CELLS	OTHERS

FBS	PPBS	B.UREA	S.CREAT	T.BILRBIN	SGOT	SGPT	ALK.PHO
				D ID			

FASTING LIPID PROFILE

T.CHO	TRIGLYCDS	HDL	LDL	VLDL

MASTER CHART

Master Chart

S. No.	AGE	WEIGHT	HEIGHT	BMI	Systolic BP	Diastolic BP	TCL	TGL	HDL	LDL=TC–(TG/5 + HDL-C)	VLDL=TG/5	BLD.UREA	S.CREATNE
1.	27	52	162	19.8	150	100	170	200	51	79	40	42	1.2
2.	21	58	157	23.53	140	90	165	195	45	81	39	21	0.6
3.	26	45	150	20	138	88	178	270	46	78	54	20	0.5
4.	22	48	165	17.63	140	88	172	220	42	86	44	28	0.9
5.	21	50	164	18.59	138	90	178	195	50	89	39	34	0.8
6.	23	56	167	20.07	142	90	162	190	49	75	38	29	0.8
7.	23	54	164	20.07	140	100	178	290	48	72	58	25	0.6
8.	25	62	149	27.92	142	98	169	198	42	87	39	33	0
9.	26	70	154	29.51	154	90	164	242	52	63	48	31	0.9
10.	30	67	153	28.62	150	96	150	200	52	58	40	24	1
11.	27	68	159	26.89	160	100	168	196	40	88	39	26	1
12.	25	84	162	32.00	142	102	150	169	43	73	33	30	1
13.	22	86	158	34.44	146	100	160	180	40	84	36	25	1
14.	26	49	167	17.56	142	96	190	290	46	86	58	22	0.6
15.	22	54	170	18.68	142	100	182	260	47	83	52	25	0.8
16.	29	65	174	21.46	152	102	198	270	52	92	54	23	0.9
17.	26	70	177	22.34	140	92	176	240	54	74	48	20	0.7
18.	22	56	148	25.56	142	90	180	200	42	98	40	18	0.9
19.	25	64	152	27.70	144	100	178	209	48	88	41	20	0.6
20.	22	48	164	17.84	160	98	170	195	51	80	39	22	0.8
21.	29	44	150	19.55	168	86	170	186	41	91	37	31	0.7
22.	25	56	154	23.61	146	102	198	290	55	85	58	23	0.9
23.	22	54	156	22.18	142	96	150	189	47	65	37	21	0.6
24.	28	57	154	24.03	140	108	169	180	45	88	36	19	0.7
25.	29	65	149	29.27	138	110	178	220	50	84	44	18	0.5

26.	23	64	164	23.79	136	112	182	260	51	79	52	30	0.9
27.	26	53	167	19.00	140	92	174	250	46	78	50	21	0.5
28.	30	50	145	23.78	142	98	181	245	47	85	49	20	0.9
29.	27	54	148	24.65	148	96	174	231	44	83	46	18	0.8
30.	22	67	165	24.60	158	100	160	200	42	78	40	22	0.7
31.	24	65	160	25.39	120	72	205	105	40	144	21	18	0.7
32.	25	66	156	27.12	116	74	199	120	42	133	24	18	0.6
33.	23	55	162	20.95	110	70	194	110	40	132	22	21	0.8
34.	26	64	165	23.50	106	68	199	106	41	136	21	22	0.9
35.	21	63	167	22.58	106	66	202	109	40	140	21	21	0.6
36.	20	62	168	21.96	120	78	195	110	39	134	22	25	1
37.	30	48	149	21.62	112	70	189	106	40	127	21	23	0.8
38.	29	45	165	16.52	110	72	195	120	38	133	24	24	0.7
39.	24	90	149	40.53	120	68	200	110	40	138	22	26	0.9
40.	25	88	167	31.55	112	74	196	116	41	131	23	30	1
41.	25	65	170	22.49	118	68	180	106	46	112	21	22	0.6
42.	22	67	158	26.83	110	68	196	105	42	133	21	18	0.9
43.	27	45	169	15.75	120	70	202	166	41	127	33	24	0.7
44.	26	87	167	31.19	120	72	182	100	46	116	20	22	1
45.	27	88	162	33.53	112	74	189	108	42	125	21	25	1
46.	27	45	162	17.14	112	72	190	132	47	116	26	24	1
47.	22	47	165	17.26	116	76	196	110	39	135	22	26	0.8
48.	23	48	154	20.23	120	74	220	112	46	151	22	23	0.6
49.	24	46	157	18.66	118	80	200	120	40	136	24	21	0.9
50.	21	56	158	22.43	116	78	199	105	42	136	21	19	0.8
51.	28	68	148	31.04	124	72	197	111	41	133	22	18	0.7
52.	24	64	157	25.96	110	70	176	122	40	111	24	21	0.6
53.	25	48	154	20.23	106	62	186	106	43	121	21	26	0.7

54.	27	44	141	22.13	100	64	190	122	44	121	24	24	0.9
55.	24	63	157	25.55	110	60	198	104	52	125	20	21	1
56.	25	78	159	30.85	120	70	192	118	43	125	23	18	0.9
57.	29	72	160	28.12	120	72	180	116	41	115	23	16	0.9
58.	20	70	176	22.59	112	68	162	111	40	99	22	16	0.8
59.	26	56	171	19.15	112	70	170	118	40	106	23	19	0.9
60.	21	44	176	14.20	100	64	188	110	41	125	22	24	0.5
61.	24	46	167	16.49	112	70	178	130	45	107	26	22	0.8
62.	26	67	165	24.60	112	68	150	101	46	83	20	26	0.7
63.	27	75	163	28.22	118	70	166	109	48	96	21	24	0.9
64.	25	56	165	20.56	114	68	182	120	43	115	24	22	0.5
65.	28	48	163	18.06	108	68	150	102	52	77	20	28	0.6
66.	25	49	165	17.99	106	70	166	103	60	85	20	18	0.8
67.	26	46	149	20.71	104	68	168	145	46	93	29	20	0.7
68.	24	52	169	18.20	104	68	170	120	48	98	24	24	0.9
69.	27	48	176	15.49	104	68	145	102	49	75	20	26	0.5
70.	24	46	175	15.02	104	70	180	100	55	105	20	18	0.8
71.	25	48	156	19.72	114	70	200	104	58	121	20	22	0.7
72.	26	55	152	23.80	106	70	168	108	48	98	21	24	0.5
73.	22	58	154	24.45	116	68	188	109	49	117	21	25	0.8
74.	23	62	158	24.83	112	78	178	110	47	109	22	21	0.9
75.	27	65	156	26.70	102	68	151	102	56	74	20	20	1
76.	23	56	165	20.56	112	68	156	108	52	82	21	24	0.9
77.	30	68	168	24.09	120	70	152	123	50	77	24	21	0.9
78.	23	63	171	21.54	100	68	162	106	47	93	21	19	0.9
79.	24	49	168	17.36	116	70	180	122	42	113	24	22	0.8
80.	25	65	169	22.75	112	60	150	103	49	80	20	21	0.7
81.	26	64	170	22.14	116	66	156	120	54	78	24	24	0.7

82.	24	67	172	22.64	118	78	154	130	58	70	26	25	0.9
83.	25	64	156	26.29	106	70	151	109	50	79	21	24	0.6
84.	24	68	157	27.58	102	68	170	101	49	100	20	20	0.5
85.	23	65	157	26.37	118	78	159	123	51	83	24	19	0.7
86.	25	65	168	23.03	106	68	163	100	57	86	20	18	0.8
87.	26	45	168	15.94	108	70	156	120	58	74	24	15	0.6
88.	22	59	158	23.63	104	60	168	104	58	89	20	19	0.5
89.	23	61	156	25.06	102	70	158	146	54	74	29	19	0.9
90.	24	80	158	32.04	112	76	180	130	48	106	26	16	0.5

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CERTIFICATE OF CLEARANCE

This is to certify that the project work titled "A COMPARITIVE STUDY OF LIPID PROFILE IN PRE-ECLAMPSIA AND NORMOTENSIVE PREGNANT & NONPREGNANT WOMEN "proposed by DR.MURALI KRISHNAN.M of K.A.P.V. Govt.Medical college, Trichy as part of fulfillment of M.D course in the subject of GENERAL MEDICINE during the academic period of 2010-2013 by The Tamilnadu Dr.MGR medical university has been cleared by the ethical committee.

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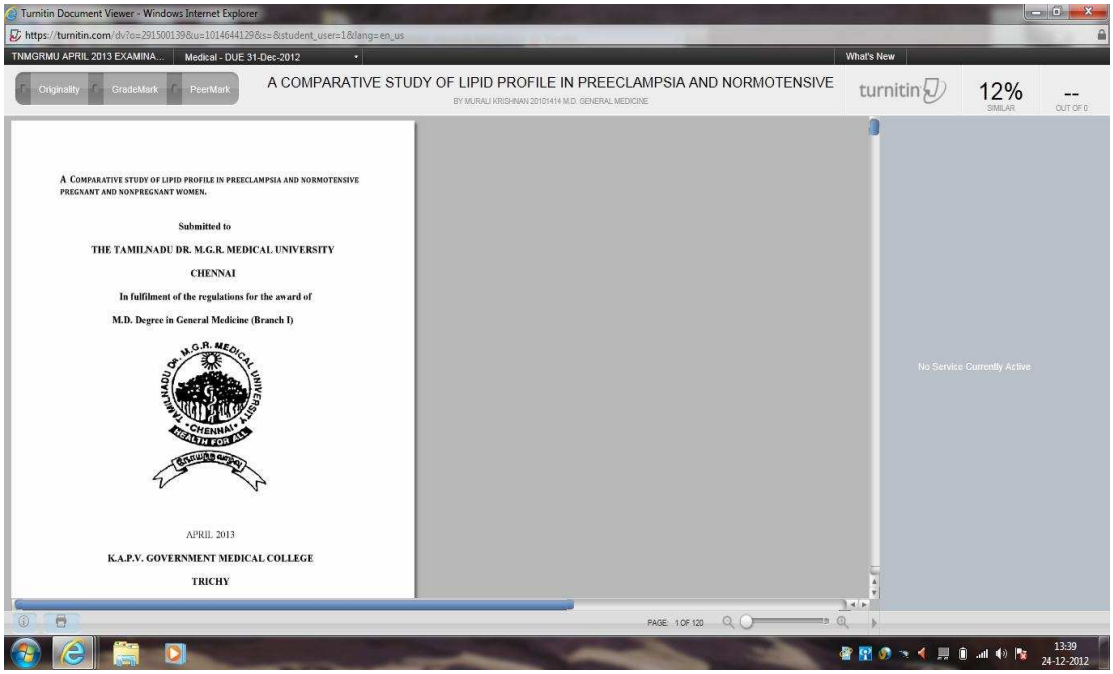
PLAGIARISM ORIGINALITY REPORT

PLAGIARISM ORIGINALITY REPORT



ABBREVIATIONS AND ACRONYMS

ABBREVIATIONS

PE	-	Preeclampsia
TGL	-	Triglyceride
TC	-	Total cholesterol
FA	-	Fatty acids
HDL	-	High density lipoprotein
LDL	-	Low density lipoprotein
VLDL	-	Very low density lipoprotein
LPL	-	Lipoprotein lipase
BP	-	Blood Pressure
SD	-	Standard Deviation